DIETARY LYSINE AND ASCITES INCIDENCE[1]

B. D. LOTT[2], S. L. BRANTON, and J. D. MAY
USDA, Agricultural Research Service, South Central Poultry Research Laboratory,
Mississippi State, MS 39762
Phone: (601) 323-2230, Ext. 204
FAX: (601) 323-3535

Primary Audience: Nutritionists, Poultry Producers

SUMMARY

Two trials were conducted with male broilers to determine the effect of dietary lysine on deaths due to ascites. Starter diets contained 1.13, 1.20, 1.28, and 1.35% dietary lysine; grower diets contained 1.02, 1.09, 1.16, and 1.23% lysine. Neither deaths due to ascites nor total deaths were affected by treatment in Trial 1. A significant effect of treatment on deaths due to ascites was observed in Trial 2, but deaths for the highest lysine level were not different from the lowest lysine level. Heart weight and ventricle weights were not affected by treatment. These results suggest that ascites is not attributable to dietary lysine levels in commercial diets.

Key words: Arginine, heart weight, temperature, ventricle weight

1997 J. Appl. Poultry Res. 6:331–334

DESCRIPTION OF PROBLEM

Losses due to ascites are of considerable concern in the production of meat-type chickens. There is widespread belief that any factor that promotes pulmonary hypertension may increase ascites incidence. The increased blood flow that occurs at low rearing temperatures increases ascites incidence. The physiological changes that lead to the development of ascites are described by Julian [1]. The underlying metabolism causing these physiological changes is not well understood. Wideman *et al.* [2] have studied arginine supplementation of the diet. Arginine is a substrate for nitric oxide and a powerful endogenous pulmonary vasodilator. Supplemental arginine reduced cumulative pulmonary hypertension syndrome mortality and lowered right:total ventricle heart weight ratios. Chickens are dependent upon dietary sources since they do not synthesize arginine at a rate sufficient to obtain maximum growth. Taylor *et al.* [3] and Dietert *et al.* [4] have shown that dietary arginine levels sufficient for maximum growth are lower than arginine levels sufficient for maximum production of nitric oxide.

The interrelationship between arginine and lysine has been well documented. Dietary lysine is directly correlated with kidney arginase activity [5]. Further, the low environmental temperatures that increase ascites incidence also cause increased plasma lysine concentration [6]. Lysine is a limiting amino

1 Trade names in this article are used solely to provide specific information. Use of trade names does not constitute a guarantee or warranty by USDA and does not signify that the product is approved to the exclusion of other comparable products.
2 To whom correspondence should be addressed

acid in typical corn-soybean meal diets. Yield of breast meat is an important economic consideration, and increasing dietary lysine has been shown to increase breast yield [7, 8]. Supplementation of the diet with 0.3% lysine has been shown to cause increased mortality of broilers exposed to cool temperatures [9].

The objective of this research was to determine whether the incidence of ascites is related to the level of dietary lysine.

MATERIALS AND METHODS

Male Ross x Ross chicks were purchased from a commercial hatchery and housed on fresh, kiln-dried pine shavings litter in two trials. Lighting was continuous and feed and water were provided *ad libitum*. In Trial 1, the rearing temperatures were: Week 1 – 29°C; Week 2 – 27°C; Week 3 – 24°C; Week 4 and following – 21°C. In Trial 2, the constant rearing temperatures were: Days 1 to 5 – 29°C; Days 6 to 14 – 26°C; Week 3 – 22°C; Week 4 – 17°C; Week 5 and following – 16°C. In each trial, 960 chicks were housed in an environmentally controlled house with 60 chicks per pen in 16 pens (1.5 × 3.65 m). Corn-soybean meal diets were formulated to meet minimum NRC [10] requirements or to exceed the requirement for lysine (Table 1). Treatments consisted of four levels of lysine in starter and grower diets. The treatments were (% lysine in starter/% lysine in grower): A – 1.13/1.02; B – 1.20/1.09; C – 1.28/1.16; and D – 1.35/1.23. To obtain the lysine levels, supplemental lysine was added to the basal starter and grower. Starter diets were fed through 3 wk of age and grower diets were fed thereafter. Feed consumption and body weights were determined at 3 wk and again at the end of each trial.

TABLE 1. Dietary composition and calculated analysis of experimental diets

INGREDIENT	STARTER	GROWER
	%	
Corn, 8.6%	58.616	62.667
Soybean Meal, 48%	32.570	28.505
Fat, 3500	4.260	4.735
Dical-Phos 22, 18.5%	2.370	2.033
Ground Limestone	1.080	1.013
Salt	0.530	0.528
Vitamin Mineral Premix[A]	0.250	0.250
Coccidostat	0.075	0.075
Lysine, 78.8%	0.004	0.000
DL-Methionine	0.245	0.194
Total	100.00	100.00
CALCULATED ANALYSIS		
Calcium, %	0.950	0.850
Available Phosphorus, %	0.480	0.420
ME/Kg	3125	3200
Arginine, %	1.500	1.372
Lysine, %	1.134	1.017
Methionine + Cystine, %	0.907	0.813
Tryptophan, %	0.287	0.262
Sodium, %	0.220	0.220

[A]The vitamin mineral premix furnished the following amounts of other ingredients per kg of feed: vitamin A, retinyl acetate, gelatin coated, 3500 IU; vitamin D_3, 1000 ICU; vitamin E, DL-α-tocopherol acetate, 4.5 IU; riboflavin, 2.25 mg; niacin, 15.0 mg; d-pantothenic acid, 4.0 mg; folic acid, 0.25 mg; vitamin B_{12}, 5 μg; choline chloride, 200 mg; thiamine, 0.5 mg; biotin, 25 μg; ethoxyquin, 12.5 mg; menadione sodium bisulfite, 1.25 mg; pyridoxine, 0.50 mg; manganese (oxide form), 24.9 mg; zinc (oxide form), 22 mg; iodine, 0.2 μg; iron (in sulfate form), 13.6 mg; and copper (in sulfate form), 1.6 mg.

Mortality was recorded as it occurred, and all dead birds were necropsied to determine cause of death. At the end of Trial 2, ventricular weight ratios were determined on 16 birds per treatment as described by Burton and Smith [11].

The data for each trial were analyzed separately with PCSAS Release 6.02 [12]. A one-way analysis of variance was used and mortality data were analyzed by Chi-square.

RESULTS AND DISCUSSION

Body weight, feed conversion, and mortality results for the two trials are given in Table 2. Left and right ventricle weight for Trial 2 is presented in Table 2. In Trial 1, ascites mortality was low. Therefore, the environmental temperature was reduced in Trial 2 in an effort to increase the incidence of ascites. Trial 2 had a greater incidence of ascites, but the incidence was not related to the level of lysine in the diet. The starter lysine level of 1.20% had a significantly lower incidence of ascites mortality than 1.13 or 1.28%, but the mortality due to ascites had no discernible relationship to dietary lysine. Moreover, dietary treatment did not significantly influence total mortality. These results do not confirm the report by Summers [9], but the highest amount of supplemental lysine in our research was 0.23%. Summers [9] supplemented diets with 0.3% lysine. There were no significant dietary treatment effects on right ventricle weights or right ventricle weight/total ventricle weight ratios. Body weights were reduced and feed conversion ratios were greater in Trial 2 than in Trial 1. This change was probably due to the lower temperatures used in Trial 2.

These data suggest that supplementing diets with lysine to improve yield will not increase ascites incidence. While the interrelationships between lysine and arginine are well documented, the physiological effects of lysine and arginine indicate that they do not affect the incidence of ascites.

TABLE 2. Effect of dietary lysine on body weight, feed gain, mortality, left ventricle weight (LV WEIGHT), and right ventricle weight (RV WEIGHT) of male broilers to 56 days of age

DIETARY LYSINE	BODY WEIGHT	FEED GAIN	ASCITES MORTALITY	TOTAL MORTALITY	LV WEIGHT	RV WEIGHT
%	g	g:g			g	g
TRIAL 1						
1.13/1.02[A]	3506	1.94	3/240	11/240[B]	n/a[C]	n/a
1.20/1.09	3552	1.94	5/240	17/240	n/a	n/a
1.28/1.16	3522	1.94	4/240	11/240	n/a	n/a
1.35/1.23	3499	1.92	2/240	13/240	n/a	n/a
TRIAL 2						
1.13/1.02	3266	2.12	11/240^{a}	19/240	9.8	3.1
1.20/1.09	3251	2.15	2/240^{b}	18/240	9.3	2.6
1.28/1.16	3284	2.12	8/240^{a}	20/240	9.4	2.7
1.35/1.23	3196	2.12	5/240^{ab}	24/240	9.4	2.9

[A]Dietary lysine in starter diet/dietary lysine in grower diet

[B]Number of deaths/number in treatment

[C]Data not collected in Trial 1

[a,b]Values without a common superscript differ significantly.

Conclusions and Applications

1. Increasing dietary lysine levels with constant dietary arginine does not increase the incidence of ascites in male broilers.
2. Increasing lysine levels to increase breast yields in broilers will not increase the incidence of ascites.

References and Notes

1. **Julian, R.J.,** 1993. Ascites in poultry. Avian Path. 22:419–454.

2. **Wideman, R.F. Jr., Y.K. Kirby, M. Ismail, W.G. Bottje, R.W. Moore, and R.C. Vardeman,** 1995. Supplemental L-arginine attenuates pulmonary hypertension syndrome (ascites) in broilers. Poultry Sci. 74:323–330.

3. **Taylor, R.L., R.E. Austic, and R. Dietert,** 1992. Dietary arginine influences Rous sarcoma growth in a major histocompatibility B complex progressor genotype. Proc. Soc. Exp. Biol. Med. 199:38–41.

4. **Dietert, R.R., K.A. Golemboski, and R.E. Austic,** 1994. Environment-immune interactions. Poultry Sci. 73:1062–1076.

5. **Jones, J.D., S.J. Petersburg, and P.C. Burnett,** 1967. The mechanism of the lysine-arginine antagonism in the chick: Effect of lysine on digestion, kidney arginase, and liver transamidinase. J. Nutr. 93:103–116.

6. **May, J.D., L.F. Kubena, F.N. Reece, and J.W. Deaton,** 1972. Environmental temperature and dietary lysine effects on free amino acids in plasma. Poultry Sci. 51:1937–1940.

7. **Moran, E.T., Jr. and S.F. Bilgili,** 1990. Processing losses, carcass quality, and meat yields of broiler chickens receiving diets marginally deficient to adequate in lysine prior to marketing. Poultry Sci. 69:702–710.

8. **Acar, N., E.T. Moran, Jr., and S.F. Bilgili,** 1991. Live performance and carcass yield of male broilers from two commercial strain crosses receiving rations containing lysine below and above the established requirement between six and eight weeks of age. Poultry Sci. 70:2315–2321.

9. **Summers, J.D.,** 1994. Ascites – A metabolic condition? Pages 199–210 in: Biotechnology in the Feed Industry. T.P. Lyons and K.A. Jacques, eds. Nottingham University Press, Loughborough, Leicestershire, UK.

10. **National Research Council,** 1994. Nutrient Requirements of Poultry. 9th Rev. Edition. Natl. Acad. Press, Washington, DC.

11. **Burton, R.R. and A.H. Smith,** 1967. Effect of polycythemia and chronic hypoxia on heart mass in the chicken. J. Appl. Physiol. 22:782–785.

12. **SAS Institute,** 1990. SAS User's Guide. Statistics. SAS Institute, Inc., Raleigh, NC.